Surgical ventricular remodeling for multiterritory myocardial infarction: Defining a new patient population

Nishant D. Patel, BA,^a Jason A. Williams, MD,^a Christopher J. Barreiro, MD,^a Pramod N. Bonde, MD, FRCS,^a Michele M. Waldron, MT, RN, CCRP,^a David C. Chang, PhD,^a David A. Bluemke, MD, PhD,^b and John V. Conte, MD^a

Conte, Patel, Barreiro, Williams, Chang, Waldron
(left to right)

Objective: Because of limited medical and surgical options for patients with end-stage congestive heart failure, we expanded the criteria for surgical ventricular remodeling to include patients with multiterritory myocardial infarction, a group historically considered high-risk candidates. We present our series of patients with multiterritory myocardial infarction who underwent surgical ventricular remodeling and propose a new patient population who may benefit from this procedure.

Methods: Data were analyzed for 51 consecutive patients undergoing surgical ventricular remodeling from January 2002 to June 2004, with 100% follow-up. Three left ventricular vascular territories were defined: anteroapicoseptal (left anterior descending), lateral (circumflex), and inferior (right coronary artery). Infarction was assessed with magnetic resonance imaging and intraoperative findings.

Results: Multiterritory myocardial infarction was found in 64.7% of patients (33/51) undergoing surgical ventricular remodeling. Mean age was 61.6 ± 11.1 years (range 40-81 years). Sixty-one percent (20/33) demonstrated evidence of myocardial infarction in all three territories. Five patients underwent concomitant mitral valve repair or replacement. Operative mortality was 6.1% (2/33) and did not differ from that of patients with single-territory infarction (11.1%, $P = .61$). Surgical ventricular remodeling significantly improved left ventricular volumes and ejection fraction in patients with multiterritory myocardial infarction. Three patients required assist device implantation, and 2 patients required defibrillator placement. Sixty-nine percent of patients in preoperative New York Heart Association functional class III or IV (22/32) had improvement to class I or II at follow-up ($P < .01$). Cox regression analysis discriminated a preoperative left ventricular end-systolic volume index greater than 100 mL/m^2 as a significant risk factor for mortality (odds ratio 12.1, 95% confidence interval 1.27-114.51, $P = .03$). Thirty-month survival of patients with multiterritory myocardial infarction ($73.5\% \pm 8.3\%$) did not differ statistically from that of patients with single-territory infarction ($n = 18$).

Conclusion: Surgical ventricular remodeling improves cardiac function and New York Heart Association functional status in patients with multiterritory myocardial infarction. Our initial results are promising and should prompt further studies to confirm our results and potentially expand the surgical ventricular remodeling inclusion criteria to include patients with multiterritory myocardial infarction.

From the Divisions of Cardiac Surgery^a and Radiology,^b The Johns Hopkins Medical Institutions, Baltimore, Md.

N.D.P. is the 2005 Chase Medical Scholar for Surgical Ventricular Restoration. J.A.W. is an Irene Piccinini Investigator, and C.J.B. is a Hugh R. Sharp, Jr, Research Fellow.

Read at the Eighty-fifth Annual Meeting of The American Association for Thoracic Surgery, San Francisco, Calif, April 10-13, 2005.

Received for publication April 13, 2005; revisions received Aug 12, 2005; accepted for publication Aug 17, 2005.

Address for reprints: John V. Conte, MD, Director of Heart and Lung Transplantation, The Johns Hopkins Hospital, Division of Cardiac Surgery, 600 North Wolfe St, Blalock 618, Baltimore, MD 21287-4618 (E-mail: jcont@csurg.jhmi.jhu.edu).

J Thorac Cardiovasc Surg 2005;130:1698-706
0022-5223/\$30.00

Copyright © 2005 by The American Association for Thoracic Surgery

doi:10.1016/j.jtcvs.2005.08.023

The prevalence of congestive heart failure (CHF) in the United States is approximately 5 million patients, and it is increasing yearly.¹ Ischemic cardiomyopathy is the cause of CHF in 3 million of these patients.¹ Approximately 35% to 50% of patients with CHF will die within 2 years without treatment.^{2,3} Although medical treatments are effective in the short term, cardiac transplantation is widely accepted as the criterion standard therapy for the treatment of CHF. Unfortunately, transplantation is limited by lack of donors, is expensive,

Abbreviations and Acronyms

CHF	= congestive heart failure
EF	= ejection fraction
IABP	= intra-aortic balloon pump
LVAD	= left ventricular assist device
LVESVI	= left ventricular end-systolic volume index
LVEDVI	= left ventricular end-diastolic volume index
MI	= myocardial infarction
MR	= mitral regurgitation
MRI	= magnetic resonance imaging
MTI	= multiterritory myocardial infarction
NYHA	= New York Heart Association
POD	= postoperative day
RESTORE	= Reconstructive Endovascular Surgery returning Torsion Original Radius Elliptical shape
STI	= single-territory myocardial infarction
SVR	= surgical ventricular remodeling

and carries many exclusions that remove it as an option for most patients. Thus, alternatives are needed for patients with advanced CHF.

Surgical ventricular remodeling (SVR) aims to ameliorate the progression of CHF by preventing further dilatation of the left ventricle, reducing myocardial wall stress, and restoring the shape and size of the chamber to improve left ventricular function. Current indications for SVR include single-territory myocardial infarction (STI); depressed ejection fraction (EF); left ventricular end-diastolic volume index (LVEDVI) greater than 100 mL/m²; left ventricular regional akinesia or dyskinesia; angina, CHF, or arrhythmias; and inducible ischemia in symptom-free patients.⁴ Relative contraindications include right ventricular hypokinesis, pulmonary hypertension, unreconstructable coronary artery disease, and myocardial infarction (MI) in multiple left ventricular territories (MTI).^{4,5}

With these criteria, results of SVR for the treatment of CHF have been excellent. Mickleborough and colleagues⁵ and Di Donato and associates⁶ have independently reported excellent 5- and 10-year survivals, with acceptable operative mortality.^{5,6} The Reconstructive Endovascular Surgery returning Torsion Original Radius Elliptical shape (RESTORE) multicenter study recently reported an operative mortality of 5.3% and an actuarial 5-year survival of 69% (22 patients at risk) in their cohort of 1198 patients with STI leading to CHF.⁷

Most authors consider MTI to be a contraindication for SVR because of the increased operative risks. However, no studies have clearly shown these patients to be at higher

risk. Because of the clinical success of SVR for STI, we expanded the criteria for SVR to include patients with MTI and present our initial results with SVR for this patient population.

Methods**Study Design**

The cases of 51 consecutive patients with SVR between January 2002 and June 2004 were retrospectively reviewed after institutional review board approval. The inclusion criterion for patients with MTI was the clear presence of infarction in two or three left ventricular vascular territories. Three regions of the left ventricle were identified by vascular supply: anteroapicoseptal (left anterior descending coronary artery), lateral (circumflex coronary artery), and inferior (right coronary artery). The presence of infarction in these territories was determined by direct observation of full-thickness scar in the opened left ventricle during surgery and was correlated with preoperative magnetic resonance imaging (MRI), cardiac catheterization, and echocardiography. MRI was the primary imaging modality used for preoperative and postoperative evaluation of left ventricular hemodynamics, wall thinning, scarring, wall motion abnormalities, shape, and volumes. Cardiac catheterization was used to determine acceptable targets for revascularization. Exclusion criteria were any of the following: absence of nonviable anterior wall myocardium, no akinetic or dyskinetic segments, minor evidence of left ventricular dilatation, poor basilar function, and unreconstructable coronary or mitral disease. The presence of MTI was not considered an exclusion criterion in this study. Thirty-three patients with MTI were included in this study, and 18 patients undergoing SVR for STI formed a comparison group.

Patient Variables

Data collection included demographic characteristics, preoperative and postoperative New York Heart Association (NYHA) functional status, hemodynamic and operative data, postoperative complications, and follow-up interviews. Mortality data were retrieved from autopsy reports, death certificates, the Social Security Death Index, and physician records. Follow-up was 100% complete, with a mean follow-up of 16.9 ± 9.4 months. Quality of life assessments were available for 23 patients with MTI who were evaluated by telephone with the Short Form 12 quality assessment tool.

Statistical Analysis

Data were collected for statistical analysis with SPSS version 11.5 statistical software (SPSS Inc, Chicago, Ill). The primary end point was overall mortality. Secondary end points included postoperative complications, the need for additional cardiac surgery, length of stay, and worsening or persistent NYHA class III or IV status. The Fisher exact test and Student *t* test were used to conduct qualitative and quantitative variable analyses, respectively. Cox regression analysis was performed to determine predictors of mortality. Descriptive statistics are presented as mean ± SD unless otherwise stated. Actuarial survival was calculated with the Kaplan-Meier method and compared by log rank analysis with that of patients who underwent SVR for STI.

Figure 1. A, Surgical plication of inferior infarct. B, Surgical plication of lateral infarct.

Operative Technique

Our operative technique has been previously described.⁸ The decision to perform SVR on the beating or arrested heart was made at the time of surgery on the basis of clamp time, concomitant procedures, and cardiac function. Our group prefers to perform SVR on the arrested heart whenever possible. In brief, after coronary artery bypass grafting and mitral valve repair or replacement, a ventriculotomy is made through the anterior myocardial wall parallel to the left anterior descending coronary artery. This incision extends distally to the apex and proximally as needed. The left ventricular walls are inspected for the presence of MI. An intraventricular sizing device (Chase Medical, Richardson, Tex; Bioventrix, Danville, Calif) is deployed into the left ventricle and is seated on the mitral annulus to aid in the reshaping process. The size of the intraventricular sizing device is based on patient body surface area in an effort to approach an optimal LVEDVI of 50 to 60 mL/m². The apex of the sizing device defines the new apex of the left ventricular chamber. A purse-string suture begins at the newly defined apex and runs along the septum, transitioning to the anterior wall. The suture is then carried down the anterior wall toward the apex along the anterior aspect of the intraventricular sizing device, outlining the new anterior wall. The tightness of the purse-string suture is determined by intraoperative surgical judgment with the intent of reducing the anterior ventriculotomy to prevent a spherical chamber. Tying the purse-string suture too tightly can result in a spherical ventricle that is excessively small. After the purse-string suture is tied, the residual defect in the anterior wall is closed with an oval Dacron polyester fabric patch if larger than 2 to 3 cm; otherwise, a linear closure is performed. Septal scarring and thinning is assessed at the time of surgery and with preoperative MRI studies. Our method of anteroapical reconstruction excludes no more than half of the height of the septum, whether a patch is used or a primary closure is performed.

Most patients with MTI underwent intraventricular balloon guided anterior wall reconstruction. The decision to plicate the lateral or inferior wall is made at the time of surgery according to the surgeon's subjective assessment of the amount of scarring and

dilatation in these territories. In patients with inferior or lateral infarcts, the scar is addressed with a linear plication. Inferior wall infarction with an interpapillary muscle distance greater than 3 cm is plicated with a suture running between the papillary muscles and ending at the apex (Figure 1, A). For patients with lateral wall infarcts, the plication suture begins at the base of the infarction and is carried to the apex (Figure 1, B). The anterior defect is then closed as previously described.

Results

Clinical Characteristics

Preoperative and intraoperative assessment of patients undergoing SVR demonstrated that 64.7% (33/51) had MTI. Of these patients, 60.6% (20/33) had a three-territory infarct, and 39.4% (13/33) had a two-territory infarct (Figure 2). The clinical characteristics of patients with MTI are presented in

Figure 2. Distribution of patients according to evidence of MI.

TABLE 1. Clinical characteristics and operative data

	MTI (n = 33)	STI (n = 18)	P value
Clinical characteristics			
Male	29 (87.9%)	14 (77.8%)	.43
White	27 (81.8%)	15 (83.3%)	>.999
Age (y, mean $\pm$ SD and range)	61.6 $\pm$ 11.1 (40-81)	64.5 $\pm$ 6.7 (48-75)	.32
Renal failure	4 (12.1%)	2 (11.1%)	>.999
Atrial fibrillation	6 (18.2%)	5 (27.8%)	.49
Previous stent or percutaneous transluminal angioplasty	10 (30.3%)	7 (38.9%)	.55
Angina	26 (78.8%)	12 (66.7%)	.52
Syncope	3 (9.1%)	5 (27.8%)	.08
Three-vessel CAD	26 (78.8%)	13 (72.2%)	>.999
Smoking	26 (78.8%)	12 (66.7%)	.5
Obesity	7 (21.2%)	2 (11.1%)	.46
Hypertension	23 (69.7%)	13 (72.2%)	>.999
Diabetes	17 (51.5%)	6 (33.3%)	.25
COPD	4 (12.1%)	4 (22.2%)	.43
Hyperlipidemia	26 (78.8%)	15 (83.3%)	>.999
Preoperative NYHA functional class			
I	0 (0%)	0 (0%)	>.999
II	1 (3.0%)	1 (5.6%)	>.999
III	14 (42.4%)	10 (55.6%)	.4
IV	18 (54.5%)	7 (38.9%)	.38
Operative data			
Mitral valve replacement	1 (3.0%)	1 (5.6%)	>.999
Mitral valve repair	4 (12.1%)	4 (22.2%)	.43
Intraventricular balloon	29 (87.9%)	16 (88.9%)	>.999
Patch reconstruction	14 (42.4%)	12 (66.7%)	.14
No. of grafts in revascularization			
5	2 (6.1%)	1 (5.6%)	>.999
4	4 (12.1%)	1 (5.6%)	.64
3	15 (45.5%)	5 (27.8%)	.25
2	7 (21.2%)	5 (27.8%)	.73
1	3 (9.1%)	0 (0%)	.54
CPB time (min, mean $\pm$ SD)	150.4 $\pm$ 61.0	125.5 $\pm$ 66.8	.18
Crossclamp time (min, mean $\pm$ SD)	85.0 $\pm$ 26.8	76.2 $\pm$ 28.1	.28
IABP	9 (27.3%)	2 (11.1%)	.29

All data are numbers of patients and percentages unless otherwise indicated. *MTI*, Multiterritory myocardial infarction; *STI*, single-territory myocardial infarction; *CAD*, coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *NYHA*, New York Heart Association; *CPB*, cardiopulmonary bypass; *IABP*, intra-aortic balloon pump.

Table 1 and did not differ significantly from those of patients with STI. Mean age was 61.6 $\pm$ 11.1 years (range 40-81) with a male/female ratio of 29:4. All patients with MTI had a diagnosis of CHF, 78.8% (26/33) had three-vessel coronary artery disease, and 78.8% (26/33) had a presentation of angina. Before the operation, 97.0% of patients with MTI (32/33) were in NYHA functional class III or IV. The remaining patient was in functional class II.

Operative Data

Operative data are also presented in Table 1 and are similar for patients with STI and those with MTI. Four patients with MTI underwent mitral valve repair, and 1 required mitral

valve replacement. Nine patients (27.3%) required intraoperative intra-aortic balloon pump (IABP) counterpulsation according to the assessment of myocardial function at the time of SVR. An intraventricular sizing device was used in 87.9% of patients (29/33), and a Dacron polyester fabric patch was used for anterior wall reconstruction in 42.4% of patients (14/33). Concomitant revascularization with an internal thoracic artery or saphenous vein was performed in 93.9% of patients (31/33). One patient who did not undergo concomitant coronary artery bypass grafting had previously undergone a stenting procedure and was therefore considered to have complete revascularization. The second patient had incomplete revascularization because of poor target vessels. This patient was in preoperative NYHA functional

TABLE 2. MRI and echocardiographic data for patients with MTI

	Preoperative	Postoperative	P value
MRI	n = 25	n = 19	
EF (%)	25.9% ± 10.7%	36.2% ± 7.7%	<.01
LVESVI (mL/m ²)	117.9 ± 72.7	68.8 ± 24.4	<.01
LVEDVI (mL/m ²)	146.4 ± 83.2	105.1 ± 34.1	.04
Echocardiography	n = 24	n = 30	
EF (%)	25.8 ± 10.7	32.6 ± 11.7	.03

All data are mean ± SD. *MRI*, Magnetic resonance imaging; *MTI*, multi-territory myocardial infarction; *EF*, ejection fraction; *LVESVI*, left ventricular end-systolic volume index; *LVEDVI*, left ventricular end-diastolic volume index.

class IV and died on postoperative day (POD) 25 of multisystem organ failure.

EF and Left Ventricular Volumes

In the MTI group, SVR significantly improved left ventricular EF and volumes on MRI (Table 2). Mean EF improved from a preoperative value of 25.9% ± 10.7% to 36.2% ± 7.7% at follow-up ($P < .01$). Mean left ventricular end-systolic volume index (LVESVI) improved from a preoperative value of 117.9 ± 72.7 mL/m² to 68.8 ± 24.4 mL/m² at follow-up ($P < .01$). Mean LVEDVI was reduced from a preoperative value of 146.4 ± 83.2 mL/m² to 105.1 ± 34.1 mL/m² at follow-up ($P = .04$). Postoperative echocardiography also demonstrated significant improvement in EF (Table 2).

When comparing patients with STI versus those with MTI, the mean preoperative EF values (29.4% ± 16.7% vs 25.9% ± 10.7, $P = .49$) and postoperative EF values (39.7% ± 14.6% vs 36.2% ± 7.7%, $P = .4$) were similar between the two groups according to MRI. Echocardiography confirmed these results, with patients with STI demonstrating a mean postoperative EF of 32.9% ± 15.7% and those with MTI having a postoperative EF of 32.6% ± 11.7% ($P = .9$). Mean preoperative EF by echocardiography also showed no difference between STI and MTI groups (20.7% ± 7.3% vs 25.8% ± 10.7, $P = .12$). Furthermore, no difference was seen between the two groups for mean postoperative LVESVI (69.3 ± 39.9 mL/m² vs 68.8 ± 24.4 mL/m², $P > .999$) or LVEDVI (108.1 ± 39.3 mL/m² vs 105.1 ± 34.1 mL/m², $P = .8$).

Mitral Valve Regurgitation

Four patients with MTI underwent mitral valve repair (ring annuloplasty), and 1 required mitral valve replacement at the time of SVR. Three patients with mitral valve repair had improvement in function with no mitral regurgitation (MR) to mild MR at follow-up. The fourth patient had no postoperative change in MR and later required left ventricular assist device (LVAD) implantation. Two patients had mod-

TABLE 3. Postoperative procedures and complications

	MTI (n = 33)		STI (n = 18)		P value
	No.	%	No.	%	
IABP	3	9.1%	2	11.1%	>.999
Atrial fibrillation	8	24.2%	4	22.2	>.999
Ventricular tachycardia	3	9.1%	1	5.6%	>.999
Junctional rhythm	1	3.0%	0	0%	>.999
Pacemaker	2	6.1%	0	0%	.53
Bleeding necessitating reoperation	1	3.0%	0	0%	>.999
Transfusion	7	21.2%	0	0%	.04
Deep vein thrombosis	5	15.2%	2	11.1%	>.999
Stroke	3	9.1%	1	5.6%	>.999
Infection					
Sternal infection	2	6.6%	1	5.6%	>.999
Pneumonia	5	15.2%	1	5.6%	.41
Urinary tract infection	4	12.1%	1	5.6%	.64
Sepsis	3	9.1%	1	5.6%	>.999
Additional surgery					
Débridement	2	6.6%	1	5.6%	>.999
LVAD placement	3	9.1%	1	5.6%	>.999
Defibrillator implantation	2	6.1%	2	11.1%	.61

MTI, Multiterritory myocardial infarction. *STI*, single-territory myocardial infarction; *IABP*, intra-aortic balloon pump; *LVAD*, left ventricular assist device.

erate to severe MR according to preoperative echocardiography, but a mitral valve procedure was not performed in these 2 cases because both patients demonstrated no MR to mild MR on intraoperative transesophageal echocardiography before SVR.

Postoperative Complications and Procedures

Postoperative complications are noted in Table 3. Complication rates were similar between the two groups, although the patients with MTI required more frequent transfusions in the postoperative period ($P = .04$). Twenty-one percent (7/33) required additional surgery, including 2 sternal wound débridements, 3 LVAD implantations, and 2 automatic implantable cardioverter defibrillator implantations in those patients with inducible ventricular arrhythmias demonstrated in the electrophysiology laboratory. After the operation, 9.1% (3/33) required IABP counterpulsation. Postoperative IABP support was indicated by hemodynamic instability and increasing intravenous inotropic agent requirements.

Mortality

Two (6.1%) operative deaths from multisystem organ failure occurred on PODs 15 and 25. This did not differ statistically from the operative mortality for patients undergoing SVR for STI at our institution (11.1%; $P = .61$). Late mortality in the MTI group was 19.4% (6/31), which did not

Figure 3. Distribution of patients with STI (anterior) versus MTI.

statistically differ from that of the STI group (6.3%, $P = .40$). The causes of late mortality in the MTI group were gastrointestinal bleeding after LVAD placement ($n = 1$), CHF ($n = 1$), sepsis ($n = 1$), multisystem organ failure ($n = 1$), LVAD-related complication ($n = 1$), and unknown causes ($n = 1$).

Actuarial survivals (including in-hospital deaths) at 6, 12, 18, 24, and 30 months were 84.3%, 78.4%, 78.4%, 73.5%, and 73.5%, respectively, which did not differ statistically from those of patients with STI (Figure 3). When patients with MTI who had LVESVI greater than 100 mL/m² were compared with those who had LVESVI less than 100 mL/m², the latter group demonstrated a statistically significant improvement in actuarial survival at 2 years ($84.8\% \pm 8.4\%$ vs $44.4\% \pm 16.6\%$, $P = .007$). Similarly, patients with LVEDVI less than 130 mL/m² demonstrated a significant improvement in actuarial survival at 2 years relative to those with LVEDVI greater than 130 mL/m² ($83.1\% \pm 9.3\%$ vs $55.6\% \pm 15.0\%$, $P = .04$).

Cox Regression Multivariable Analysis

Cox regression analysis was performed to determine independent predictors of mortality in the MTI group. Variables used in the analysis included age older than 60 years, chronic obstructive pulmonary disease, incomplete revascularization, recent MI (≤ 30 days), diabetes, three-territory MI, NYHA functional class IV, LVESVI greater than 100 mL/m², preoperative renal failure requiring dialysis, pulmonary hypertension, preoperative atrial fibrillation, and previous stenting. Of these variables, LVESVI greater than 100 mL/m² resulted in a 12-fold increase in mortality ($P = .03$, 95% confidence interval 1.27-114.51).

Functional Outcomes

SVR significantly improved functional outcomes for patients with MTI (Figure 4). Before the operation, 97.0% of

Figure 4. Preoperative (light bars) versus postoperative (dark bars) NYHA functional class for patients with MTI undergoing SVR. Asterisk indicates $P < .05$.

patients (32/33) were considered to be in NYHA functional class III or IV; this had improved to 30.3% (10/33) at follow-up ($P < .01$). Sixty-nine percent of patients (22/32) with MTI in preoperative NYHA functional class III or IV had improvement to class I or II at follow-up ($P < .01$).

Short Form 12 telephone questionnaire data, available for 23 patients with MTI, indicated that 69.6% (16/23) reported their general health as good to excellent after SVR. Furthermore, 78.3% (18/23) of patients with MTI indicated little to no limitation in physical activities after the procedure.

Discussion

SVR is an established treatment for ischemic cardiomyopathy resulting from STI. Revascularization performed concomitantly with SVR improves left ventricular size, EF, and NYHA functional class in patients with anterior akinetic or dyskinetic scar^{5-7,9-11} and improves regional myocardial performance in remote nonischemic areas.^{12,13}

Despite this success, patients with CHF resulting from MTI have been denied SVR because these patients were believed to face a greater operative risk. Previous studies by Menicanti and colleagues⁴ and Mickleborough and colleagues⁵ have described operative techniques for managing large apical aneurysms by plicating the posterior wall. Some of these patients may have had MTI, but the populations were not clearly defined, and outcomes in patients with MTI have not been specifically addressed. In a recent study, Mickleborough and colleagues⁵ stated that SVR was not contraindicated for patients with gross chamber dilatation and extensive wall motion abnormalities. We believe that postinfarction enlargement does not reflect the territory of MI. Many patients in our study had markedly dilated left ventricles with extensive wall motion abnormalities and still demonstrated STI on intraoperative assessment and preop-

erative MRI studies. Similarly, an early report from Di Donato and associates⁶ described 14 patients undergoing SVR with anterior wall MI and remote asynergy. That study, however, provided no conclusive evidence (intraoperative observation of full-thickness scarring or MRI data) to demonstrate MTI in those 14 patients. We specifically evaluated outcomes after SVR in patients with confirmed MTI. We saw significant improvements in EF, left ventricular volumes, and NYHA functional status, with acceptable survival for these high-risk patients.

Some centers use the transition zone between contractile and noncontractile myocardium, among other factors, to determine the margins of anterior wall reconstruction⁵; others use the base of the papillary muscles as a guide.⁹ In patients with diffuse disease and MTI, this transition zone can be difficult to recognize. In this setting, and for patients with large ventricles, we have found commercially available intraventricular sizing devices to be extremely helpful. For patients who have enlargement in two or more territories, these sizers can act as templates for plication of the inferior and lateral walls. This allows the restoration of normal chamber geometry while excluding as much fibrous scar as possible.

We saw significant improvement on MRI in EF, LVESVI, and LVEDVI. These results are similar to those previously reported by centers evaluating outcomes after SVR for STI.^{5-7,9-11} We recognize that MRI is not as readily available as echocardiography when assessing cardiac function. Echocardiography is therefore commonly used to measure left ventricular dimensions, EF, and mitral valve function when MRI is unavailable. However, echocardiography clearly cannot provide the sensitivity and specificity that MRI allows. We recommend MRI when available for assessing cardiac function for patients undergoing SVR. MRI is especially important for assessing left ventricular function in patients with MTI, because the lateral and inferior walls can be visualized to assess dilatation and thinning.

We performed a subset analysis of outcomes for two-versus three-territory MI but did not find any differences in survival or cardiac function. However, these inconclusive findings may result from a type II error. Our early experience with patients undergoing SVR for MTI leads us to believe that lateral wall MI may be a predictor of worse outcome, because we believe that the lateral wall contributes more to the function of the left ventricle than does the inferior wall. We hope to further analyze the impact of two-versus three-territory MI and the presence of lateral versus inferior MI in future studies.

Recent reports have demonstrated that left ventricular volumes are a significant predictor of outcome. White and associates,¹⁴ Yamaguchi and colleagues,¹⁵ and the recent Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries trial¹⁶ each demonstrated poorer survival

for patients with LVESVI greater than 60 mL/m². Our study demonstrated 42% and 28% decreases in LVESVI and LVEDVI, respectively, after SVR. Additionally, our patients with MTI who had LVESVI greater than 100 mL/m² faced a significant increase in mortality relative to those who had LVESVI less than 100 mL/m². Our LVESVI cutoff for increased risk of death was higher than previously reported,¹⁴⁻¹⁶ but this can be explained by a significantly higher average LVESVI in our patient population than in those in previous studies.⁵⁻⁷ In addition, increasing the number of patients in our cohort would likely have reduced this LVESVI threshold.

MR is common among patients with ventricular enlargement because annular dilatation and chordal restriction inhibit effective leaflet coaptation.¹⁷ In our series, 4 patients with MTI underwent mitral valve repair concomitantly with SVR, and 1 patient required mitral valve replacement. Improved left ventricular geometry after SVR can reduce the degree of MR; however, surgery may also worsen papillary muscle or chordal restriction in some cases. Intraventricular sizing devices may help to avoid this complication, as can be seen in our series, in which none of our patients demonstrated worsening of mitral valve function. Our current recommendations include the use of intraventricular sizing devices where appropriate and a concomitant mitral valve procedure in any patient with worse than mild MR.

In our cohort, 3 patients with MTI required LVAD placement at 3, 9, and 35 days. All 3 of these patients had three-vessel coronary artery disease and a preoperative EF no greater than 20%, with a history of MI 4 days, 2 weeks, and 1 year before SVR. Two patients died in the hospital, one on POD 60 of complications from rectal cancer and the other on POD 129 of LVAD-related complications. The final patient who required LVAD implantation died at 18 months from massive gastrointestinal bleeding. From our experience, we believe that the anatomic consequences of SVR do not preclude patients from future LVAD implantations if necessary. We therefore discuss LVAD implantation with patients who fulfill the accepted criteria in the event that SVR fails to improve cardiac function.

Our 30-month actuarial survival of 73.5% ± 8.3% for patients undergoing SVR for MTI is not statistically different from our survival of patients undergoing SVR for STI (82.5% ± 9.2%). Other studies have demonstrated excellent survival after SVR. Mickleborough and colleagues⁵ reported a 30-month survival of approximately 85% for 174 patients with CHF undergoing SVR, which was significantly worse than the survival for patients without CHF in the same study (n = 111). They also found that CHF and preoperative EF lower than 20% were predictors of poor 5-year outcome on multivariable analysis. The recent RESTORE multicenter study⁷ reported 30-month survivals of approximately 85% for patients in preoperative NYHA

functional class III and 65% for patients in functional class IV. In the same RESTORE study, patients with mean preoperative EF lower than 30% and LVESVI greater than 80 mL/m² had 30-month survivals of approximately 80% and 75%, respectively. Our initial experience with SVR for patients with CHF and MTI, who had mean preoperative EF of 26% and LVESVI of 118 mL/m², demonstrates encouraging early and midterm clinical results despite severe left ventricular dysfunction and markedly dilated left ventricular volumes before the operation.

Although we have demonstrated that patients with CHF and full-thickness MI in multiple left ventricular vascular territories are candidates for SVR, there clearly must be some amount of viable myocardium remaining. However, this amount has yet to be determined. Our belief is that the base of the heart is most critical, and so we offer SVR to candidates with MTI who retain basilar function. We are able to visually assess basilar function with MRI, but the current technology does not permit us to quantify these findings. We hope that future advances in MRI technology will allow us to determine the function and viability of individual left ventricular segments quantitatively and thus enable the cardiac surgeon to identify appropriate candidates for SVR.

Our study is limited by retrospective analysis, a small patient cohort, and a relatively short follow-up interval. Even with these limitations, however, we were able to demonstrate similar survivals, improvements in cardiac function, and improvements in quality of life for patients with MTI and STI. Longer follow-up and a larger patient cohort will enable us to make better long-term conclusions about SVR for patients with MTI, but our initial results are quite promising.

References

1. Roger VL, Weston SA, Redfield MM, Hellermann-Homan JP, Killian J, Yawn BP, et al. Trends in heart failure incidence and survival in a community based population. *JAMA*. 2004;292:344-50.
2. Mills RM Jr, Young JB, editors. Practical approaches to the treatment of heart failure. Philadelphia: Williams & Wilkins; 1998. p. 2.
3. Carson PE. Beta blocker treatment in heart failure. *Prog Cardiovasc Dis*. 1999;41:301-21.
4. Menicanti L, Di Donato M. The Dor procedure: what has changed after fifteen years of clinical practice. *J Thorac Cardiovasc Surg*. 2002;124:886-905.
5. Mickleborough LL, Merchant N, Ivanov J, Rao V, Carson S. Left ventricular reconstruction: early and late results. *J Thorac Cardiovasc Surg*. 2004;128:27-37.
6. Di Donato M, Toso A, Maioli M, Sabatier M, Stanley A, Dor V, et al. Intermediate survival and predictors of death after surgical ventricular restoration. *Semin Thorac Cardiovasc Surg*. 2001;13:468-75.
7. Athanasuleas CL, Buckberg GD, Stanley AW, RESTORE Group. Surgical ventricular restoration in the treatment of congestive heart failure due to post-infarction ventricular dilation. *J Am Coll Cardiol*. 2004;44:1439-45.
8. Conte JV. Surgical ventricular restoration: techniques and outcomes. *Congest Heart Fail*. 2004;10:248-51.
9. Maxey TS, Reece TB, Ellman PI, Butler PD, Kern JA, Tribble CG, et al. Coronary artery bypass with ventricular restoration is superior to coronary artery bypass alone in patients with ischemic cardiomyopathy. *J Thorac Cardiovasc Surg*. 2004;127:428-34.
10. Di Donato M, Sabatier M, Montiglio F, Maioli M, Toso A, Fantini F, et al. Outcome of left ventricular aneurysmectomy with patch repair in patients with severely depressed pump function. *J Am Coll Cardiol*. 1995;76:557-561.
11. Dor V, Sabatier M, Di Donato M, Montiglio F, Toso A, Maioli M. Efficacy of endoventricular patch plasty in large postinfarction akinetic scar and severe left ventricular dysfunction: comparison with a series of large dyskinetic scars. *J Thorac Cardiovasc Surg*. 1998;116:50-9.
12. Di Donato M, Sabatier M, Toso A, Barletta G, Baroni M, Dor V, et al. Regional myocardial performance of non-ischemic zones remote from anterior wall left ventricular aneurysm. *Eur Heart J*. 1995;16:1285-92.
13. Di Donato M, Barletta G, Maioli M, Fantini F, Coste P, Sabatier M, et al. Early hemodynamic results of left ventricular reconstructive surgery for anterior wall left ventricular aneurysm. *Am J Cardiol*. 1992;69:886-90.
14. White HD, Norris RM, Brown MA, Brandt PW, Whitlock RM, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. *Circulation*. 1987;76:44-51.
15. Yamaguchi A, Ino T, Adachi H, Murata S, Kamio H, Okada M, et al. Left ventricular volume predicts postoperative course in patients with ischemic cardiomyopathy. *Ann Thorac Surg*. 1998;65:434-8.
16. Migrino RQ, Young JB, Ellis SG, White HD, Lundergan CF, Miller DP, et al. End-systolic volume index at 90 to 180 minutes into reperfusion therapy for acute myocardial infarction is a strong predictor of early and late mortality. The Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO)-I Angiographic Investigators. *Circulation*. 1997;96:116-21.
17. Bolling S. Mitral valve reconstruction in the patient with heart failure. *Heart Fail Rev*. 2001;6:177-85.

Discussion

Dr Lorenzo A. Menicanti (*San Donato, Italy*). SVR is a well-established procedure that represents a useful surgical tool. It was proposed at the beginning for classic aneurysmal dilatation of the left ventricle after acute anteroapical MI. With increased experience, the procedure has been applied to a larger spectrum of pathology, particularly to real postischemic cardiomyopathy, and this study is a clear demonstration. It is evident, as pointed out here by Patel and colleagues, that to be successful a critical amount of working myocardium necessary to sustain the systemic circulation must be present at the end of the procedure. So it is not only a problem of the number of territories involved but mainly of extension of the infarction and consequently of quality and quantity of remaining tissue. This article described 33 patients with dilated postischemic cardiomyopathy determined by MTI. These patients represent a very difficult cohort with increased surgical risk.

I have three questions for you, Dr Conte. First, according to your experience and your MRI analysis, what contraindication criteria have you adopted in your clinical practice?

Second, we know that even after a successful procedure with good reduction of left ventricular volume, a few patients are seen 4 to 6 months later with an increase in left ventricular dimension. Do you have the same results, and if so, do you have some predictive criteria for this evolution?

Third, mitral insufficiency plays an important role in this group of patients, and when it is addressed, the mortality is increased. In your article, you describe a few cases in which mitral insufficiency disappeared after SVR alone. Can you comment on this and define your strategy for this challenging subgroup of patients?

Dr Conte. Dr Menicanti, thank you for those insightful comments. I acknowledge your contribution to this field; without you

and Dr Dor having led the way, I think we would not have this field at all.

First, all procedures have contraindications, and I think this is a difficult group in which to identify absolutes, but certainly there is some quantifiable amount of muscle that must remain for us to be able to successfully perform this operation. I don't think we have identified that yet, but I am optimistic that with MRI techniques and like technologies we will be able to quantify the exact amount of muscle necessary to support the circulation, or at least to determine the amount that will lead to operative failure. So I think the only patients I would not consider are those patients who have little to no basilar function retained, because in my experience those are absolutely the patients who do not do well, and you have to have at least one or preferably two basilar segments remaining to have a successful outcome.

Second, you asked how long to wait after an infarction to perform this operation. The video we showed nicely demonstrated very thick scar tissue, and that makes the operation somewhat easier and certainly a pleasure to perform, but it is not always there. So I would recommend waiting at a minimum 3 months, preferably 6 months if you can. This operation is best performed for chronic rather than acute heart failure. Some of the patients in our series came to light because of acute infarction, but as we evaluated these patients preoperatively, they clearly had evidence of old infarctions, and that was the indication for remodeling in these patients with acute infarctions. So certainly I would wait 6 months, if possible.

The final question had to do with MR, and I have actually significantly changed my approach to these patients. My goal is to leave the operating room with no MR in these patients, if possible. A few patients who have not had mitral repairs performed have still had a reduction in the amount of MR. It seems to me that the reason for that is that, just like in the diagram I showed for the inferior plication, the papillary muscles are brought closer together and probably toward the mitral annulus a little bit. That may reduce some of the restriction on those papillary muscles and chords, decreasing the tethering and allowing those leaflets to coapt a little bit better. I think that is the mechanism for the successful reduction in MR.

Conversely, when we do this operation we run the risk, particularly when we put our purse-string too close to the papillary muscles, of restricting the papillary muscles and chords even more,

worsening the MR. So it is certainly a sensitive situation sometimes.

Dr Robert Guyton (*Atlanta, Ga*). I congratulate you on the series. The question I have is regarding the use of the IABP. I find these patients to be ideal candidates for prophylactic use of an IABP. As you are aware, a couple of prospective series have shown that for these high-risk patients, prophylactic use of the IABP decreases intensive care unit and hospital stays. Particularly because we can now put the IABPs in with a relatively low risk if we use transesophageal echocardiographic guidance and survey of the descending aorta, would this group of patients not be an ideal group for prophylactic use of an IABP?

Dr Conte. Absolutely; I couldn't agree more. Ever since we began doing this operation several years ago, that is the exact approach we have taken. Every patient has an arterial line placed in the groin in preparation for an IABP. If I went back and actually really looked at those patients who received an IABP, approximately a third of the total, I would tell you that those are the patients with extremely poor function, with larger ventricles. We have an extremely low threshold for putting in the IABPs, and we keep them in until we are able to begin vasodilators in the intensive care unit. Many of these patients require postoperative vasodilators and in fact will even be transferred to the step-down unit or the floor with a regimen of milrinone or like agents.

Dr Radu Deac (*Targu-Mures, Romania*). I congratulate you for the excellent results you obtained with a difficult group of patients. We have a much smaller series of patients, all of whom survived the operation. The long-term results of this group were not as good for all of them. I have one question. What is the rule to design the size of the patch, since the patch remains an akinetic area in the anterior ventricular wall?

Dr Conte. That is a good question, but I don't think there is any good answer. The practice that I have taken up is really just to go and look at it, and if it is larger than about 3 cm, I patch everybody. One of the risks I think you do run is if you have a long anterior ventriculotomy and you put a large purse-string in there and then you do not patch it, as you close that anterior ventriculotomy you are going to pull on the papillary muscles, restrict leaflet motion a little bit, and worsen the MR. So I think it is particularly important in patients with very large ventricles or those who have had extremely proximal anterior infarctions to place a patch.